

Hyperkalaemia: Management of Non-Oliguric Hyperkalaemia in the Preterm Infant

Key Points

1. If there are:
 - a. no ECG abnormalities, confirm hyperkalaemia on non-haemolysed arterial or venous specimen
 - b. Peaked T waves or arrhythmia on ECG:
 - i. Calcium gluconate 10% 0.11mmol/kg (0.5mL/kg over 10-20minutes).
 - ii. Sodium bicarbonate 4.2% 2mmol/kg over 10-20minutes (not simultaneously via the same line).
2. Discontinue any IV fluids containing K⁺ (including PG1).
3. Commence insulin (0.1units/kg/hour) with Glucose 25% (250micrograms/kg/hour) via a central or umbilical line (refer to Insulin protocol in IV medicine manual). Gradually reduce insulin and glucose (I/G) over hours to prevent rebound hypoglycaemia and hyperkalaemia.
4. Correct concomitant hypocalcaemia (calcium correction).
5. Investigate and treat underlying cause of acidosis.
6. Avoid oral and rectal resonium.

1. Purpose

Hyperkalaemia [serum potassium (K⁺) >7mmol/L] has been reported in up to 50% ELBW premature infants. It may be associated with ECG disturbances (prolonged atrioventricular and ventricular conduction) and arrhythmias (sinus bradycardia or ventricular tachycardia).

Ninety-eight percent (98%) of total body K⁺ is intracellular at a concentration of 150mmol/L, with the extracellular concentration 30 times lower.

Hyperkalaemia may result from:

- increased K⁺ uptake
- decreased K⁺ excretion
- shift of K⁺ from the intracellular to the extracellular space due to immature function of the erythrocyte Na⁺/K⁺ - ATPase. This is the aetiology in non-oliguric hyperkalaemia in the preterm infant in the first few days after birth which is unrelated to leakage following cell disruption associated with hypoxia, acidosis, hypoglycaemia, bruising, intracranial haemorrhage or haemolysis.

This guideline outlines the requirement for the management of Non-Oliguric Hyperkalaemia in the Preterm Infant at the Women's.

2. Definitions

Hyperkalaemia: Serum K⁺ > 7mmol/L in non-haemolysed arterial or venous blood.

It is not uncommon for K⁺ to be greater than 7mmol/L on a capillary specimen. Therefore, hyperkalaemia should always be confirmed with a non-haemolysed arterial or venous specimen.

3. Responsibilities

Staff caring for a preterm Infant with Hyperkalaemia should follow this guideline.

4. Guideline

4.1 Treatment

In general, serum K⁺ > 7mmol/L is well tolerated in preterm infants.

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Hyperkalaemia should be treated when:

- $K^+ > 7\text{mmol/L}$ *AND ventricular arrhythmia *
- $K^+ > 7.5\text{mmol/L}$ *AND peaked T waves confirmed on 12 lead ECG
- $K^+ > 8\text{mmol/L}$ *

(*non-haemolysed arterial or venous blood)

Treatment of hyperkalaemia aims to:

1. reduce the likelihood of arrhythmias associated with hyperkalaemia
2. redistribute K^+ into the intracellular space
3. remove K^+ from the body.

The same principles apply to the treatment of oliguric renal failure (in consultation with the paediatric renal physician at the Royal Children's Hospital, Parkville).

Recommended treatments

1. If there are:
 - a. no ECG abnormalities, confirm hyperkalaemia on non-haemolysed arterial or venous specimen
 - b. Peaked T waves or arrhythmia on ECG:
 - i. Calcium gluconate 10% 0.11mmol/kg (0.5mL/kg over 10-20minutes).
 - ii. Sodium bicarbonate 4.2% 2mmol/kg over 10-20minutes (not simultaneously via the same line).
2. Discontinue any IV fluids containing K^+ (including PG1).
3. Commence insulin (0.1units/kg/hour) with Glucose 25% (250micrograms/kg/hour) via a central or umbilical line (refer to Insulin protocol in IV medicine manual). Gradually reduce insulin and glucose (I/G) over hours to prevent rebound hypoglycaemia and hyperkalaemia.
4. Correct concomitant hypocalcaemia (calcium correction).
5. Investigate and treat underlying cause of acidosis.
6. Avoid oral and rectal resonium.

Consider the following important information

1. Calcium ions antagonize the membrane effects/ arrhythmogenicity of hyperkalaemia.
2. Acidosis may increase hyperkalaemia by shifting potassium from the intra- to extracellular space.
3. Cation-exchange resin (resonium) does not effectively reduce serum K^+ in non-oliguric hyperkalaemia in preterm infants. In addition, complications of rectal ion exchange resins include impaction and/or rectal perforation.
4. Insulin and glucose (I/G) decreases serum K^+ by transporting potassium into the intracellular space. In a small retrospective study, I/G reduced K^+ to $<6.5\text{mmol/L}$ within 5hours. When I/G was compared with cation-exchange resin, there was a trend towards reduced all cause mortality [RR 0.18 (95% CI 0.03, 1.15)], duration of hyperkalaemia and the incidence of IVH \geq grade 2 [RR 0.30 (95% CI 0.10, 0.93)].
5. Salbutamol is a β -adrenergic agonist that causes cellular potassium uptake by stimulating membrane bound $\text{Na}^+/\text{K}^+ - \text{ATPase}$. It is frequently used to treat hyperkalaemia in older children and adults, but there is limited evidence to support its use in the preterm infant.
 - a. Infusion: 100nanogram/kg/min over 24hours or 4-5microgram/kg over 15-20 minutes.
 - b. Inhalation: When compared with nebulised saline, serum K^+ was reduced at 4hours and at 8hours after initiation of treatment. Inhaled salbutamol appeared to be well tolerated, but potential side effects

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include tachycardia, hypertension, tremor and hyperglycaemia.

6. Exchange transfusion may be considered as a last resort where all other therapeutic options have failed. However, a long preparation time is required to use the recommended saline washed RBC.

7. Peritoneal dialysis may also be considered as a last resort.

The Cochrane review 'Interventions for non-oliguric hyperkalaemia in preterm neonates' included 3 RCTs comprising 74 preterm infants and concluded that no firm recommendations for clinical practice could be made. Treatment with I/G is preferred over treatment with rectal cation-resin, but both I/G and salbutamol inhalation deserve further study. No serious adverse effects were noted with either I/G or salbutamol inhalation.

5. Evaluation, monitoring and reporting of compliance to this guideline

Compliance to this guideline will be monitored, via clinical incident reported through Victorian Health Incident Management System (VHIMS).

6. References

1. Vemgal P, Ohlsson A. Interventions for non-oliguric hyperkalaemia in preterm neonates. Cochrane Database of Systematic Reviews 2012, 16 May 2012 | DOI: 10.1002/14651858.CD005257.pub3
2. O'Hare FM, Molloy EJ. What is the best treatment for hyperkalaemia in a preterm infant? Arch Dis Child. 2008;93:174-176
3. Mildenberger E, Versmold HT. Pathogenesis and therapy of non-oliguric hyperkalaemia of the premature infant. Eur J Pediatr. 2002;161:415-422

7. Legislation/Regulations related to this guideline

Not applicable

8. Appendices

Not applicable.

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